

Postmenopausal Women and their Bone Health

Qureshi AS*, Sarmad Rehan and Khizar Hayat

Department of Anatomy, University of Agriculture, Faisalabad Pakistan

***Corresponding author:** Anas Sawar Qureshi, Department of Anatomy, University of Agriculture, Faisalabad Pakistan, E-mail: anas-sarwar@uaf.edu.pk

Mini Review

Volume 1 Issue 2 Received Date: September 18, 2017 Published Date: September 26, 2017

Bones make much important work in our body. It not only creates a body structure but also protects vital organs (like heart, brain, lungs) within our body [1]. The medullary cavity present in bone is involved in the production of blood cells as well as cells like hepatocytes and fibroblasts under specific conditions [2]. The inorganic calcium ions present in the bone regulate the serum level of calcium in the body [3]. Bones help us in movement as well as keep our body upright so we can move around. If there is any abnormality in the bones or in the joints, it hinders our ability to move around freely [4]. An adult human being has 206 bones present in the body which are linked to one another with the help of cartilage, tendon, and ligaments.

The modern era has brought many facilities to the human beings which has led to rather a slackness in behavior and lack of will to do hard physical work [5]. In addition to this change in behavior, improper diet has also played an important role in the spread of diseases like diabetes and osteoporosis [6]. According to the studies conducted on the prevalence of osteoporosis, it was estimated that almost 200 million people are suffering from this disease and the prevalence is still on the rise. Women are found to be more prone to this disease especially after the age of 40 years. The incidence rate increases from 5%-29% during the age of 60-79 years while the change is only 5%-13% in case of males during the same period [7].

What is osteoporosis?

Osteoporosis is a disease in which the osteum (wall of bone) becomes very thin, intercellular space increases thus the density of the bone is decreased which leads to increased fracture risk [2]. This is the biggest cause of bones in older people. The vertebral column, hip joint, and wrist joint are most likely quintessential targets of developing osteoporosis [8]. Not only fracture but osteoporosis may also lead to malformation of bones which later on causes pain due to the compression of surrounding muscle tissue [9]. After the induction of menopause in elderly women, rate and progression of osteoporosis increase dramatically due to the reduction in the serum level of estrogen [10].

Usually, girls are 14 years old and maturity of boys at the age of 18. Maturity remains at the age of 30 years of age. After that, the bone remodeling starts. During this, parts of the old bones naturally break and the new parts are done in those places. Some cabbage breaks and causes factors to occur at the same time. Cellular bone cells are called bone breeding cells, and the cells that end up the old bone are called bone cells (Osteoclast). Stem cells are both osteocytes that are called 'mature' cells.

Whenever the body undergoes through remodeling, the number of osteoblasts increases, and the number of osteoclasts decreases, which improves the bone formation and decreases the bone resorption procedure. Although it is a dynamic process it is kept in balance by the release of factors like IGF etc. [11]. Osteoporosis results due to hyperactivation of osteoclasts and decrease in the functioning of osteoblasts which leads to imbalance. This causes a reduction in the capability of the body to form new bone tissue and thus the pre-existing ones become porous and weak [12].

In females malfunctioning thyroid gland and back to back pregnancies has proven to be a supplementary factor for the induction of osteoporosis. Pregnancy in elderly women (35-40 years) especially leads to relatively more cases of osteoporosis than in young ones [13]. Estrogen has been established as an effective component for preventing the bone resorption via the suppressing activity of the osteoclasts [14]. After menopause, reduction in levels of progesterone and estrogen occurs thus giving the osteoclasts an opportunity to become active which leads to increase in bone destruction [15].

Journal of Human Anatomy

The ratio of annual waste in bones in our body is 1 to 2.24 percent, but the rate increases by 2 to 4 percent when age is 50 years or older. However, in some people, this rate can be up to 8 percent. After 50 years the menupause occurs and if normal health is not good, the disease of osteoporosis is obsolete.

Types of Osteoporosis

- Primary Osteoporosis
- Secondary Osteoporosis II
- Primary Osteoporosis

Primary osteoporosis is further classified as:

Post menopause: This closure occurs during the period. Bones in this type of osteoporosis are very weak due to which chances of bone breakdown increase [16].

Senile: This type is the one which results due to aging. It may occur in individuals with age of more than 70 years irrespective of the gender [17].

Secondary Osteoporosis

Osteoporosis is caused by any deficiency or illness, such as unbalanced diet, lack of hormones, orthopedic cancer, or due to excessive use of pediatric medicine, is called secondary osteoporosis [18]. The major reason for the onset of osteoporosis is the deficiency of calcium in the diet. Calcium is mainly obtained by the body from the food but if it has low quantities of calcium then body gets calcium from the bone due to increased activity of osteoclasts as a compensatory mechanism [19]. In addition to the amount of calcium, other inorganic ion like phosphorus also plays an important role. The ratio of calcium and phosphorus is as much important as the total quantities of these ions [20].

Symptoms of Osteoporosis

The synonym used for this disease is "thief disease" as the person suffering from this condition stays unknown to its progression until most of the calcium has already been removed from the bony tissue [21]. At this point, due to the removal of the large quantity of calcium, bones become thin like egg shells and break instantly even on the lightest nudge.

Women with osteoporosis suffer from wrist fracture in ratio 1:3 while the proportion of males is 1:6 [6].

Radiating pain from the sacral region of the backbone towards the legs [22].

Extreme pain in chest bones (like ribs and sternum) [23]. Bone fractures on slight nudge or injury [24].

Suffering individuals look younger than their actual age due to a malformed bony structure.

The rate of disease in women globally is high as compared to males. The ratio is higher in light-colored societies while the lowest disease rate is recorded in individuals belonging to African countries.

Pre-disposing Factors

Among the different risk factors for osteoporosis, the following are the most important:

Drinking is alcohol the most important cause of osteoporosis. High consumption of alcohol can lead to leaching of calcium ions from bony tissue which leads to decrease in density of bone and it becomes more vulnerable to fracture [25,26].

Ovariectomy leads to the dramatic decrease in serum estrogen level which causes hyperactivation of osteoclasts [27].

Diseases like the decrease in appetite, malfunctioning of thyroid and kidney diseases can also act as predisposing factors for osteoporosis [28].

Unhealthy habits like smoking and insufficient exercise are also reported to be causing factors of osteoporosis. Smoking cause decrease in activity of bone forming cells (osteoblasts) and also cause breakdown of estrogen in case of females [29].

Certain medicines (like proton pump inhibitors) can also lead to decrease in the amount of calcium ions in the bones [30].

Lack of Vit. D causes an increase in the functioning of the parathyroid gland. This gland produces calcitonin which causes bone resortion. The decrease of Vit. D in the body may be due to an insufficient exposure to sunlight [31].

Malnutrition of important inorganic ions (like calcium, phosphorus, sodium, iron etc) and Vitamins (like Vit. A, D, C and K) in children are important for proper deposition of calcium in bones. Deficiency of these microelements can cause osteoporosis in later life [32].

Soft drinks containing phosphoric acid can cause chelation of free calcium ions, as well as removal of calcium from bony tissue, thus causing a decrease in strength of bones [33].

Journal of Human Anatomy

It is interesting to note that there are only a few reported cases of this metabolic disease in those individuals who are more inclined towards high body score.

Precautions

- Avoid lifting heavy weights especially in improper posture.
- > Wear short-heeled shoe and walk with straight back.
- Use calcium-rich food like milk to fulfill the requirements of calcium in the body. Make some time to sit in the sunlight so as to fulfill the Vit. D deficiency.
- Avoid smoking and drinking.
- > Perform regular exercise.

Treatment

Bisphosphonates can be used to prevent bone fractures in osteoporosis patients [34]. If they are used for 3-4 years it can be more beneficial and bone fracture risk can be reduced by 52 to 57 percent [35]. The risk of a fracture is very much reduced after administration of bisphosphonates orally for five years and intravenously for three years [36]. In case of higher risk per, oral medication can be continued for up to ten years and intravenous for up to six years [37]. However, there are reports available about the potential side effects of longterm use, therefore the longer duration of therapy with bisphosphonates should be discouraged [38].

Flouride supplements increase the bone density but after menopause, they are not beneficial and can't reduce the damage caused by fracture [39]. Some studies report that in a postmenopausal woman, strontium therapy can help prevent spinal damage [40]. Hormonal therapy is one of the most effective choices of treatment in postmenopausal women suffering from osteoporosis [41,42]. Raloxifene has been effective in reducing effects of osteoporosis upon the backbone [43]. However, it doesn't have compensatory effects on other bones. Denosumab also proves useful in treating osteoporosis [44,45].

References

1. Kronenberg HM (2003) Developmental regulation of the growth plate. Nature 423(6937): 332-336.

- 2. Bianco P, Riminucci M, Gronthos S, Robey PG (2001) Bone marrow stromal stem cells: Nature, biology, and potential applications. Stem cells 19(3): 180-192.
- 3. Peacock M (2010) Calcium metabolism in health and disease. Clin J Am Soc Nephrol 5(Suppl 1): S23-S30.
- Cappozzo A, Catani F, Leardini A, Benedetti MG, Croce UD (1996) Position and orientation in space of bones during movement: Experimental artifacts. Clin Biomech (Bristol, Avon) 11(2): 90-100.
- 5. Tiwari AK (2011) Diabetes: Time to look beyond gluttony and laziness. Indian J community med 36(4): 253-258.
- 6. Hofbauer LC, Brueck CC, Singh SK, Dobnig HJ, Kanis (2006) An estimate of the worldwide prevalence and disability associated with osteoporotic fractures. Osteoporosis international 17(12): 1726-1733.
- 7. Reginster JY, Burlet N (2006) Osteoporosis: A still increasing prevalence. Bone 38(2): 4-9.
- 8. Cummings SR, Melton LJ (2002) Epidemiology and outcomes of osteoporotic fractures. Lancet 359(9319): 1761-1767.
- 9. Silverman SL (1992) The clinical consequences of vertebral compression fracture. Bone 13: S27-S31.
- 10. Khosla S, Melton LJ, Riggs BL (2011) The unitary model for estrogen deficiency and the pathogenesis of osteoporosis: Is a revision needed? J Bone Miner Res 26(3): 441-451.
- 11. Phan TC, Xu J, Zheng MH (2004) Interaction between osteoblast and osteoclast: Impact in bone disease. Histol Histopathol 19(4): 1325-1344.
- Boyle WJ, Simonet WS, DL Lacey (2003) Osteoclast differentiation and activation. Nature 423(6937): 337-342.
- 13. Khovidhunkit W, Epstein S (1996) Osteoporosis in pregnancy. Osteoporosis international 6(5): 345-354.
- 14. Nakamura T, Imai Y, Matsumoto T, Sato S, Takeuchi K, et al. (2007) Estrogen prevents bone loss via estrogen receptor α and induction of Fas ligand in osteoclasts. Cell 130(5), 811-823.

- 15. Prior JC (1990) Progesterone as a bone-trophic hormone. Endocr Rev 11(2): 386-398.
- 16. Hernandez CJ, Beaupré GS, DR Carter (2003) A theoretical analysis of the relative influences of peak BMD, age-related bone loss, and menopause on the development of osteoporosis. Osteoporos Int 14(10) 843-847.
- 17. Duque G, Troen BR (2008) Understanding the mechanisms of senile osteoporosis: New facts for a major geriatric syndrome. J Am Geriatr Soc 56(5): 935-941.
- 18. Harrop JS, Prpa B, Reinhardt MK, Lieberman (2004) Primary and secondary osteoporosis' incidence of subsequent vertebral compression fractures after kyphoplasty. Spine 29(19): 2120-2125.
- 19. Garriguet D (2011) Bone health: Osteoporosis, calcium and vitamin D. Health Rep 22(3): 7-14.
- 20. Heaney RP (2004) Phosphorus nutrition and the treatment of osteoporosis. Paper presented at the Mayo Clinic Proceedings 79(1): 91-97.
- 21. Kamienski M, Denise T, Vega M (2011) The silent thief: Diagnosis and management of osteoporosis. Orthop Nurs 30(3): 162-171.
- 22. Chung SK, Lee SH, Kim DY, Lee HY (2002) Treatment of lower lumbar radiculopathy caused by osteoporotic compression fracture: The role of vertebroplasty. J Spinal Disord Tech 15(6) 461-468.
- 23. Sapherson DA, Mitchell SC (1990) Atraumatic sternal fractures secondary to osteoporosis. Clin Radiol 42(4): 250-251.
- 24. Raisz LG (2005) Pathogenesis of osteoporosis: Concepts, conflicts, and prospects. J Clin Invest 115(12): 3318-3325.
- 25. Kanis JA, Johansson H, Johnell O, Oden A, De Laet C, et al. (2005) Alcohol intake as a risk factor for fracture. Osteoporos Int 16(7) 737-742.
- Sampson HW (1997) Alcohol, osteoporosis, and bone regulating hormones. Alcohol Clin Exp Res 21(3): 400-403.
- 27. Lasota A, Danowska-Klonowska D (2004) Experimental osteoporosis-different methods of

ovariectomy in female white rats. Rocz Akad Med Bialymst 49(1): 129-131.

- 28. Cunningham J, Sprague SM (2004) Osteoporosis in chronic kidney disease. American journal of kidney diseases 43(3): 566-571.
- 29. Zhao LJ, Liu YJ, Liu PY, Hamilton J, Recker RR, et al. (2007) Relationship of obesity with osteoporosis. J Clin Endocrinol Metab 92(5): 1640-1646.
- Targownik LE, Lix LM, Metge CJ, Prior HJ, Leung S, et al. (2008) Use of proton pump inhibitors and risk of osteoporosis-related fractures. Canadian Medical Association J 179(4): 319-326.
- 31. Weaver CM, Alexander DD, Boushey CJ, Dawson-Hughes B, Lappe JM, et al. (2016) Calcium plus vitamin d supplementation and risk of fractures: An updated meta-analysis from the national osteoporosis foundation. Osteoporosis International 27(1): 367-376.
- 32. Bianchi ML (2013) Causes of secondary pediatric osteoporosis. Pediatr endocrinol Rev 10(2): 424-436.
- 33. Peters BS, Martini LA (2010) Nutritional aspects of the prevention and treatment of osteoporosis. Arq Bras Endocrinol Metabol 54(2): 179-185.
- McClung M, Harris ST, Miller PD, Bauer DC, Davison KS, et al. (2013) Bisphosphonate therapy for osteoporosis: Benefits, risks, and drug holiday. Am J Med 126(1): 13-20.
- 35. Body JJ (2011) How to manage postmenopausal osteoporosis? Acta Clin Belg 66(6): 443-447.
- 36. Fleisch HA (1997) Bisphosphonates: Preclinical aspects and use in osteoporosis. Ann Med 29(1): 55-62.
- Watts NB, Diab DL (2010) Long-term use of bisphosphonates in osteoporosis. The Journal of Clinical Endocrinology and Metabolism 95(4): 1555-1565.
- 38. Kwek EB, Goh SK, Koh JS (2008) An emerging pattern of subtrochanteric stress fractures: A long-term complication of alendronate therapy? Injury 39(2): 224-231.
- 39. Riggs BL, Hodgson SF, O'Fallon WM, Chao EY, Wahner HW, et al. (1990) Effect of fluoride treatment on the

Journal of Human Anatomy

fracture rate in postmenopausal women with osteoporosis. New England Journal of Medicine 322(12): 802-809.

- 40. Reginster JY, Felsenberg D, Boonen S, Diez-Perez A, Rizzoli R, et al. (2008) Effects of long-term strontium ranelate treatment on the risk of nonvertebral and vertebral fractures in postmenopausal osteoporosis: Results of a five-year, randomized, placebo-controlled trial. Arthrits Rheum 58(6): 1687-1695.
- 41. Grady D, Rubin SM, Petitti DB, Fox CS, Black D, et al. (1992) Hormone therapy to prevent disease and prolong life in postmenopausal women. Ann Intern Med 117(12): 1016-1037.
- 42. Lane NE, Sanchez S, Modin GW, Genant HK, Pierini E, et al. (1998) Parathyroid hormone treatment can reverse corticosteroid-induced osteoporosis. Results of a randomized controlled clinical trial. J Clin Invest 102(8): 1627-1633.

- 43. Delmas PD, Ensrud KE, Adachi JD, Harper KD, Sarkar S, et al. (2002) Efficacy of raloxifene on vertebral fracture risk reduction in postmenopausal women with osteoporosis: Four-year results from a randomized clinical trial. J Clin Endocrinol Metab 87(8): 3609-3617.
- 44. Steven R Cummings, Javier San Martin (2009) Denosumab for prevention of fractures in postmenopausal women with osteoporosis. N Engl J Med 361(8): 756-765.
- 45. Lasota A, Danowska-Klonowska D (2011) The effect of vitamin D on bone and osteoporosis. Best practice and research Clinical endocrinology and metabolism 25(4): 585-591.